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CASE REPORT

High-frequency oscillatory ventilation (HFOV) facilitates CO_2 elimination in small airway disease: The open airway concept

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Introduction

The use of high-frequency oscillatory ventilation (HFOV) in pulmonary conditions with increased airway resistance and prolonged time constants, such as small airway disease (SAD), remains controversial, in particular if an underlying airway abnormality like tracheobronchomalacia is present. This is based upon the assumption that with this mode of ventilation the risk of dynamic air trapping is increased.[1] However, this case report demonstrates that HFOV can be successfully used as an early rescue therapy in these patients by applying a technique aimed at opening and stenting the airways.[2]

Case

A 21 month old boy with tracheobronchomalacia was mechanically ventilated for bronchiolitis with respiratory failure. His chest radiograph revealed hyperinflation with bronchial thickening compatible with bronchiolitis. Disease course was complicated by increasing hypercapnia despite increasing inspiratory pressures and lowering respiratory rates to facilitate prolonged expiration. On day 4, the PAP was 31 cm H_2O and the arterial $PaCO_2$ 108 mmHg (Fig. 1). The chest radiograph showed progressive hyperinflation, infiltrates were absent. Oxygenation also became impaired despite an increase of FiO_2 to 0.8 and positive end-expiratory pressure of 9 cm H_2O. We then started HFOV (Sensormedics 3100 A, Yorba Linda CA, USA) rescue therapy. Initial settings were continuous distending pressure (CDP) 15 cm H_2O, proximal pressure amplitude (ΔP) 65, inspiration time 33%, frequency 6 Hz, continuous flow 20 l/min, FiO_2 0.6. Figure 2 depicts the course of $PaCO_2$, PaO_2 and HFOV settings. Initially the $PaCO_2$ decreased, but quickly increased probably from pulmonary overdistension. We lowered the CDP to 13 cm H_2O, but this did not have any effect on the $PaCO_2$ and PaO_2 (with decreasing the CDP, a rise in the PaO_2 was expected). Overdistension was unlikely, and we hypothesized that the small airways were still occluded. We subsequently used the CDP to open and stent the small airways to facilitate gas

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exchange. The frequency was decreased to 5 Hz to increase tidal volume and the CDP was increased incrementally. With increasing CDP, the $PaCO_2$ gradually decreased, and after 10 h of HFOV at CDP 28 cm H_2O, the child could be normoventilated. We concluded that at a CDP of 28 cm H_2O, all occluded small airways were opened. To prevent barotrauma, we carefully lowered the CDP and ΔP and still could normoventilate the child. During HFOV therapy, dopamine (max 10 mcg/kg/min) was continuously infused to maintain an adequate blood pressure, as this became partially impaired due to high intrathoracic pressure. Diuresis did not get impaired (minimum > 1.5 ml/kg/h). He was on HFOV for 7 days and subsequently successfully switched to conventional ventilation. After 4 days, conventional ventilation could be discontinued.

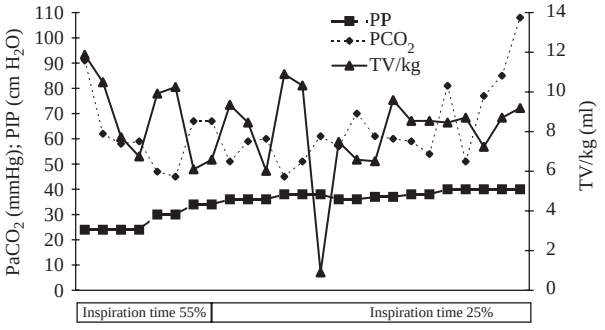

Figure 1 Settings on conventional ventilation before initiation of HFOV.

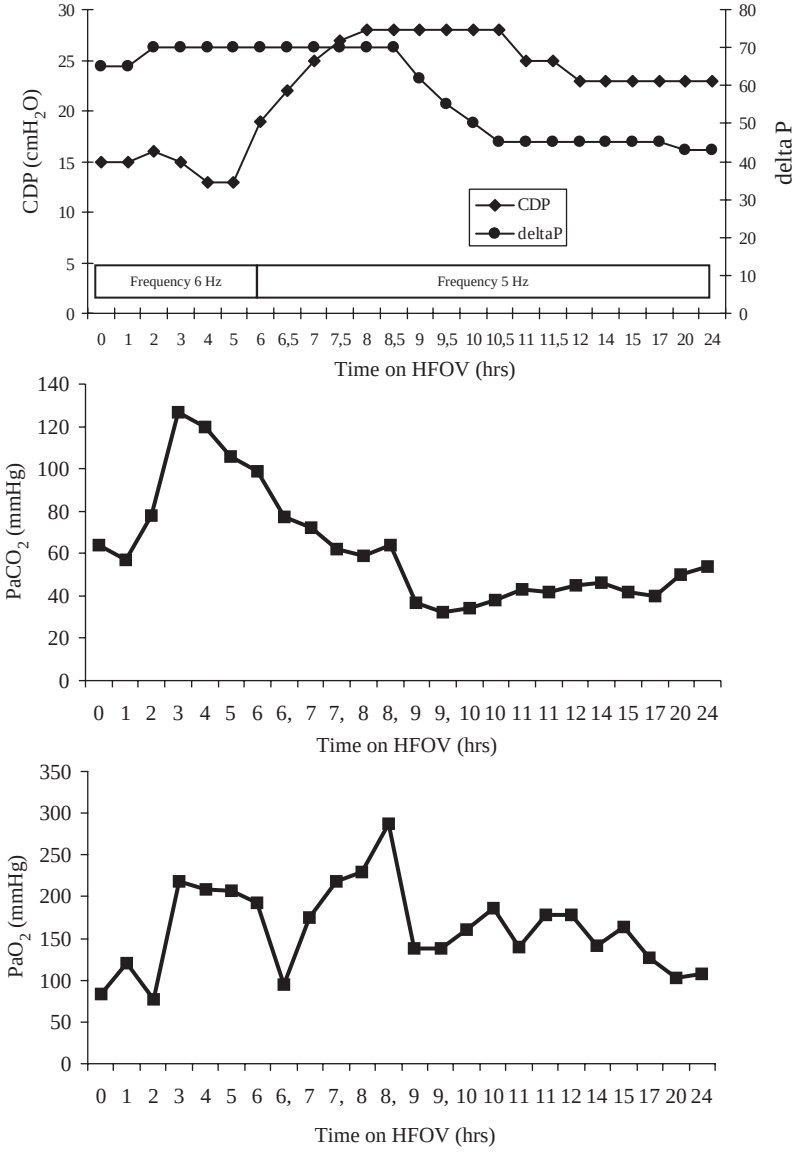

Figure 2 Time course of HFOV settings (CDP and ΔP) and results from arterial blood gas analysis ($PaCO_2$ and PaO_2).

Discussion

In this patient, HFOV was successfully used as a rescue therapy for refractory acidosis due to incomplete alveolar ventilation resulting from inadequate alveolar emptying in SAD. The CO_2 clearance was further decreased due to pre-existing bronchomalacia. In our situation, the CDP was used to open up and stent the small airways (''open airway'' concept in analogy to the ''open lung'' concept) to provide adequate ventilation which is in sharp contrast with the application of CDP to provide optimal oxygenation.[2] Our experience supports the thrifty evidence available that HFOV can be considered in SAD.[3]

In SAD, both inflammation of the bronchial wall with oedema and epithelial necrosis, as well as bronchial hyperreactivity, contribute to airflow limitation. This means that the hysteresis of these airways is altered.[4,5] Because of the increased muscular tone, higher pressures are needed to open up the small airways and prevent them from collapsing. On conventional ventilation, the airways close during passive expiration and re-open during inspiration in SAD. This re-opening requires high pressures and the airways are exposed to large pressure swings, which are unwanted in the perspective of lung-protective ventilation. With HFOV, the airways are opened up and remain open with a CDP that is constant and not influenced by pulmonary regional differences in time constants. The superimposed oscillations can move freely in and out the alveoli providing adequate ventilation with low pressure swings as the oscillations are attenuated in the bronchial tree.[6] The benefit of active expiration on the Sensormedics 3100 is that air is being actively removed to clear CO_2 without the risk of air trapping.[7]

The approach we describe should only be used as a rescue therapy and if one is experienced with HFOV. For instance, the CDP must be applied carefully and stepwise; if the airways are opened up, compliant alveoli can be faced with high pressures. Every incremental change should be followed by $PaCO_2$ determination to see at which CDP the airways are opened and the $PaCO_2$ decreases. This opening of peripheral airways is time-dependent since the pressure is propagated through the bronchial tree in a stepwise manner; this explains the gradual decrease in $PaCO_2$ in our patient.[8] When the airways are open, the lowest possible CDP and ΔP should be sought to minimize the risk of overdistension, permissive hypercapnia is allowed if airway patency is restored. Application of high pressures with CDP result in high intrathoracic pressures; this means that inotropic support and vascular fillings might be necessary to maintain adequate blood pressure and diuresis.

It could be argued that with high PEEP on conventional ventilation, a similar effect would have been achieved. However, achieving the mean airway pressure necessary to open the airways probably resulted in higher peak inspiratory pressures with large pressure swings, which must be avoided because of the chance for barotrauma.

In conclusion, we show that in SAD rescue therapy with HFOV with active expiration can be successfully employed to open airways and facilitate gas exchange.

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